Amendments to the Specification:

Please replace the first paragraph on page 1 with the following amended paragraph:

This application claims the benefit of U.S. Patent Application Serial No. 09/907,411 filed July 17, 2001, now abandoned and U.S. Patent Application Serial No. 60/218,895 filed July 18, 2000, the disclosure of which are incorporated herein by reference.

Please delete the paragraphs starting at page 1, line 16 through line 20, on page 2, which is the section titled "Brief Description of the Drawings".

Please replace the paragraph beginning at page 47, line 21, with the following amended paragraph.

Genomic sequences flanking transgenic Ht12 DNA were isolated from the events TC1 and TC0 by using a plasmid-rescue approach. As explained earlier, the events were generated by particle bombardment in Hi-II background and crossed to a Pioneer inbred line PHN46. Several self-ligated clones from the NsiI or EcoRI digested transgenic DNA survived over antibiotic selection from both events. Two clones, designated as 19-2 and 28, were respectively obtained from TC1 and TC0 and were selected for further analysis. The clones 19-2 and 28 were about 5 Kb and 3.2 Kb long, respectively. Restriction enzyme analysis confirmed that the original cloning sites were retained in the clones. Further analysis revealed that at least one restriction site that was not in either of the two co-transformed plasmids was present in the clones ~~(Figure 1)~~. This strongly suggested the existence of a stretch of non-plasmid DNA sequence in the clones selected.

Please replace the paragraph beginning at page 48, line 6, with the following amended paragraph:

To verify that the Ht12 was originally transformed into a Hi-II chromosome in TC1, RFLP profiles of Hi-II, PHN46, and TC1 were compared ~~(Figure 2)~~. DNA samples from those lines were digested with the restriction enzyme *Bam*HI and probed with a 700 bp non-plasmid DNA fragment isolated from the clone 19-2. Hi-II and PHN46 each showed a single hybridized band but the Hi-II's hybridized fragment (about 7 kb) was much larger than that of PHN46 (about 1.0 kb). Two hybridization fragments were observed in TC1. One of them was equivalent in size to that of PHN46 while the other was smaller than that of Hi-II (6.5 kb vs. 7 kb). No corresponding Hi-II fragment was observed in TC1. These results suggested that the Ht12 gene in TC1 must have originally integrated into a Hi-II chromosome, which agreed with the known fact described above.

Please replace the paragraph beginning at page 48, line 17, with the following amended paragraph:

The transgenic DNA on a Hi-II chromosome in TC0 was confirmed through SNP analysis. The SNP profiles in the genomic regions flanking the transgenic DNA among Hi-II, PHN46, and TC0 ~~are presented in Figure 3~~ were obtained. A total of 6 SNPs were observed between Hi-II and PHN46 in the amplified PCR products. The sequencing profile of TC0 was the same as that of Hi-II, confirming that the transgenic DNA was integrated into a Hi-II chromosome in TC0.

Please replace the paragraph beginning at page 48, line 29, with the following amended paragraph:

Inverse-PCR technique was used to clone genomic sequences flanking transgenic *mi1ps* expression cassettes in Hi-II X PHN46 F1 hybrids. Sequences contiguous to the right borders of T-DNA were obtained from more than 10 *mi1ps* events. In most cases, a second PCR was necessary to obtain specific amplification of a desired PCR product ~~(Figure 4)~~. The length of flanking sequences cloned from the study varied, ranging from below 200 bp up to more than 1 kb long.

Please replace the paragraph beginning at page 49, line 7, with the following amended paragraph:

Parental origins of transgenic chromosomes in 6 *mi1ps* events (Table 8) were determined, two by Southern blot and four by SNP analyses, in this study. ~~Figure 5 shows the~~ The hybridization patterns of Hi-II, PHN46, and the F1 transgenic *mi1ps* event 2482.53-1-12A were determined. The DNA samples were digested by the restriction enzyme NheI and the blot was probed with a flanking sequence isolated from the transgenic event 2482.53-1-12A. Hi-II showed two hybridized bands (1.4 and 0.6 kbs) that were also present in the F1 transgenic hybrid 2482.53-1-12A. Three hybridization bands were observed in PHN46 and one of them (0.6 kb) was also present in both Hi-II and 2482.53-1-12A. The two other PHN46 bands (1.5 and 1.1 kbs) were not present in 2482.53-1-12A. Instead, they were replaced by a novel band (6.0 kb) in 2482.53-1-12A. These results suggest that the *mi1ps* gene in 2482.53-1-12A integrated into a PHN46 chromosome.

Please delete the paragraph beginning at page 50, line 1, which starts with "An example of determining".

Please delete Figures 1, 2, 3, 4a, 4b, 5, 6 and 7.